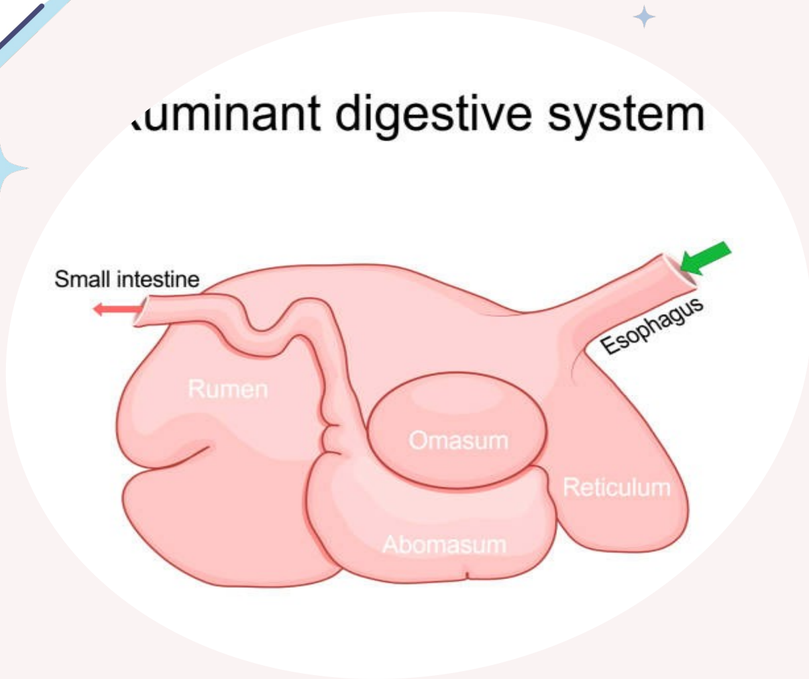
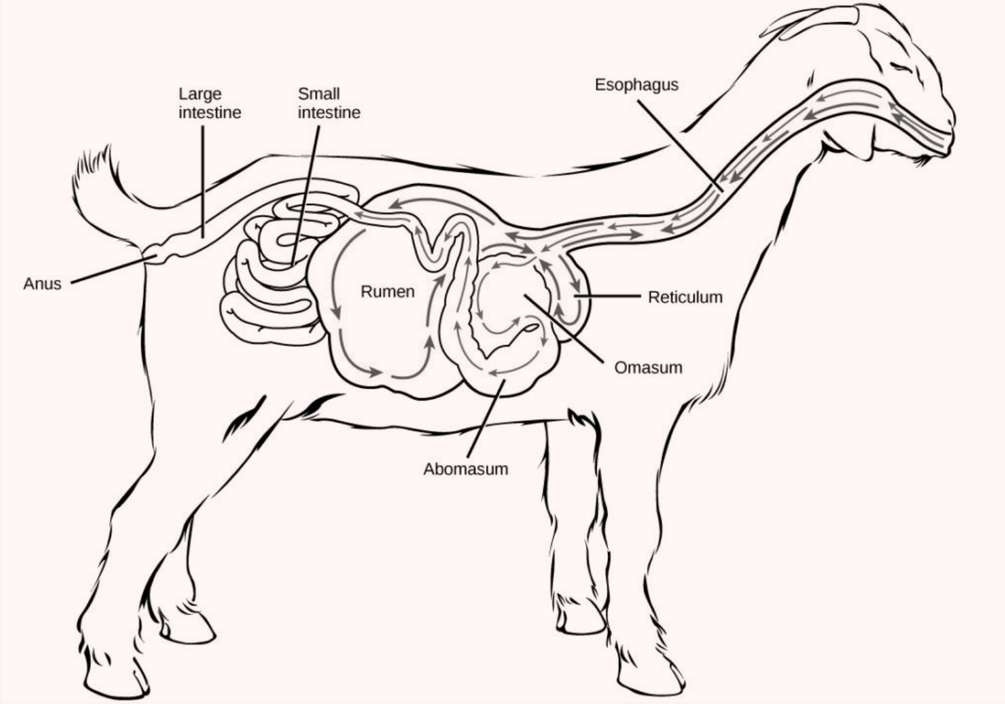
Diseases of compound stomach





Vagal indigestion

Houfler’s syndrome

chronic indigestion

- **Definition :**

Chronic disease affecting forestomach & abomasum of ruminants due to lesions involving **vagus nerve** resulting in varying degrees of paralysis of stomach

CCC by :

- **Anorexia**, dehydration, weakness, weight loss & milk production
- Gradual distention of **left paralumbar fossa primarily** then
- **Bilateral distension** of **ventral** abdomen (due to inability of passage of ingesta)
- Delayed passage of ingesta soft, pasty, dark, scanty foul odour feces and no defecation.

NB : when vagus nerve is affected , first noticed change is : ↑ in tone of nerve whether this lesion is due to TRP,... etc ↑ in tonicity of nerve appearance of disease . So disease appears primarily from ↑ of tonicity of vagus nerve. (↑ weak impulses)

Vagal indigestion, Houfler’s syndrome, chronic indigestion

Etiology :

The etiology of vagus indigestion has been controversial but has been divided into 2 major subcategories as a **complication for TRP :**

1. Vagal nerve injury

- Dorsal vagal nerve injury achalasia of reticulo-omasal orifice (anterior stenosis)
- Ventral vagal nerve injury achalasia of pylorus (posterior stenosis)

2. Reticular fibrinous adhesions

In TRP , During chronic peritonitis adhesion between reticulum and peritoneum or other organs, these adhesions could affect rumen motility

- **Ventral branch supplies abomasam and pylorus**

?

in addition, there are some other causes.

- Ruminal, reticular, abomasal **walls** diseases as inflammation,ulcer,peritonitis.
- Abomasal displacements, dilatation, torsion & ulcer or thrombosis of its vessels.
- After surgical correction of abomasum esp. when surgical correction is delayed
- Bacterial → actinobacillosis
- Parasitic → sarcosporidiosis & cysticercus tinuicollis larvae
- peritonitis in sheep
- Generally any conditions affect vagus

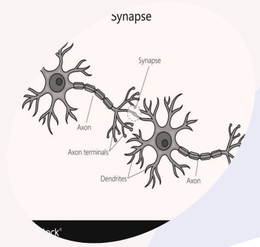
nerve pathway along

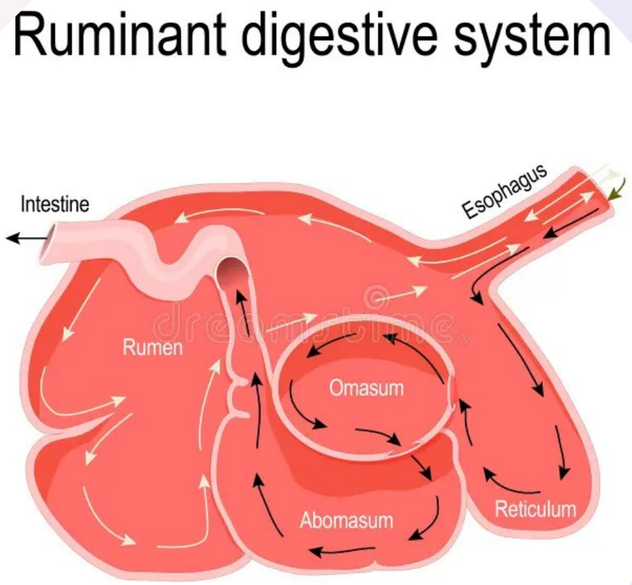
Vagal indigestion, Houfler’s syndrome, chronic indigestion

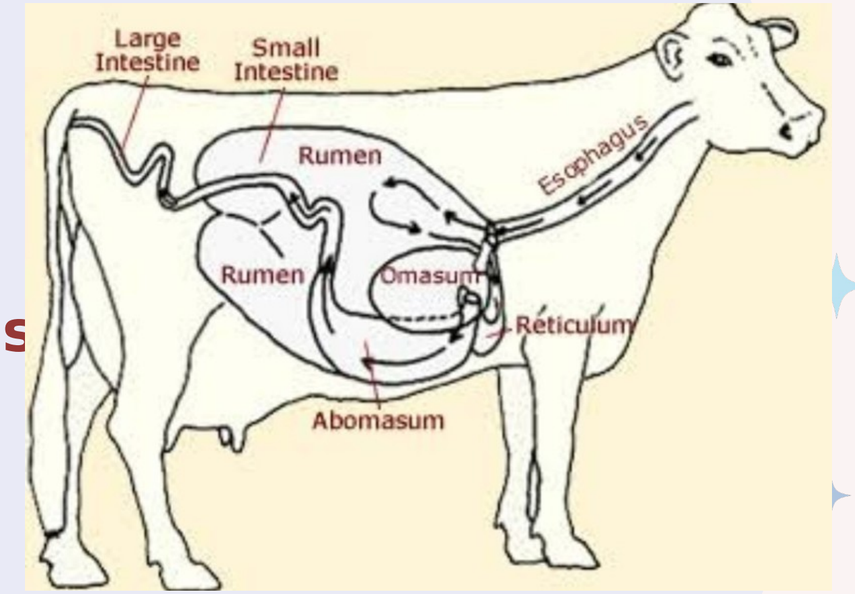
Pathophysiology:

Obstruction of ingesta flow occurs at **two** sites:

- **Anterior functional stenosis (achalasia):** passage of ingesta from the ruminoreticulum to the omasum & abomasum is impaired, resulting in rumen overload.





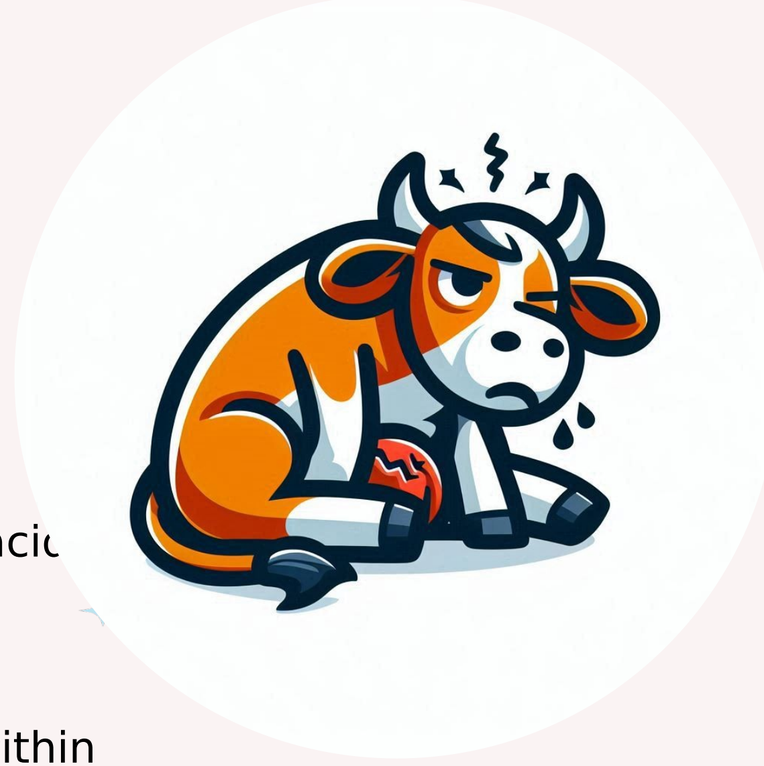


Posterior functional stenosis (achalasia):

Abomasal emptying is impaired, leading to abomasal overload & reflux of **abomasal** contents into the **omasum** & **ruminoreticulum**.

this case is more dangerous , it’s glandular stomach therefore during accumulation of food continuous secretion of hcl loss of cl inside abomasum contents & K **metabolic**

Vagal indigestion, Houfler’s syndrome, chronic indigestion



Clinical signs : **Vagus indigestion were classified into 3 types :**

General symptoms of 3 forms:

- **Inappetence** for several days or complete anorexia with loss of B.wt & milk
→ended by inanition (exhaustion caused by lack of nourishment) , dehydration, acid base imbalance, weakness ,recumbency & death finally without response to treatment.

- **Physical parameters (pulse,temp..),** vital signs & response to stimuli still within normal range on the first 2 weeks of case until late stages.

- **Abdominal inspection (esp in anterior stenosis:** enlargement in **upper** left flank (in late stages)(apple shape) due to accumulation of ingesta and eventually enlargement of **lower right** flank (pear shape)

Both enlargements papple shape or L shape

- **Ruminal palpation** (from external & rectal) :

Mushy to watery consistency

this is because when food is trapped rumen stratification is lost and content is porridge like

- **Defecation:**

Vagal indigestion, Houfler’s syndrome, chronic indigestion

Clinical signs :

Vagus indigestion were classified into 3 types :

- **Related to anterior stenosis**

Ruminal distension with hypermotility

This form is not related to pregnancy or parturition.
Moderate to **severe recurrent bloat** is common.
1) Rumen inspection (ripples or undulation) (weak contractions with ↑ frequency , rate)
2) Rumen auscultation &ballottement (↓ intensity) & abnormal sounds（**fluid splashing sound** or **ping sound** ） during auscultation and percussion)
3) Heart rate (normally vagus nerve has inhibitory action on heart so ↑ in tonicity ） bradycardia : 44 bpm, normally 60-80
heart sounds ） systolic murmur (due to heart compression） displacement of valve)

Ruminal distension with atony

Common in late pregnancy & may persist after calving.

- Gross distension of abdomen & rumen that may block pelvic cavity during rectal examination due to sever distension.
- Rumen auscultation: (weak intensity & absence of ruminal sounds abnormal sounds : same as hypermotility)
- The animal loses weight rapidly, becoming weak & recumbent
- At this stage : the heart rate increases markedly : tachycardia)
- The animal dies slowly due to inanition, dehydration, electrolyte
- acid-base imbalances.

- **Related to posterior stenosis**

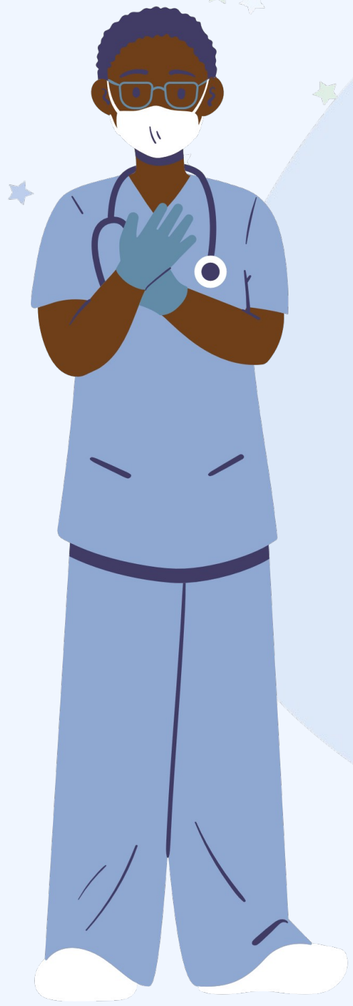
Pyloric obstruction & abomasal impaction

Most cases occur in late pregnancy

- Inspection : abdominal distension: **may not appear**, depends on degree of fermentation
- Rumen auscultation: absence of ruminal movements
- Abomasum inspection, palpation:
during first stages : distention at right lower flank (doesn’t appear at pregnancy, first stages. Per rectum may reach abomasum which is normally not reached during rectal palpation
- Abomasal auscultation (abnormal sound) : fluid splashing sound , ping at right lower flank
- Terminal stages: most dangerous ） acid base ,electrolytes imbalance ） continuous secretion of hcl ） loss of H & cl ions ） ↑ cl , h, k ions in ruminal content ） **Hypochloremic hypokalemic metabolic alkalosis.**
- reflux of abomasal content to rumen may happen which is rich in cl ions which is toxic to microflora ） ↑

Vagal indigestion, Houfler’s syndrome, chronic indigestion

Laboratory evaluation



1. Proximal functional **stenosis** laboratory findings are **nonspecific**.
2. **Distal stenosis** is ass. with abomasal reflux syndrome **hypochloremia , hypokalemia, while ↑ cl in ruminal fluid metabolic alkalosis**
3. **Hypocalcemia, hypoglycemia** in early, mild cases due to malnourishment , ↓ in feed intake
4. **↑ Total protein** due to dehydration & fibrinogen concentrations (due to adhesions in cases of TRP)
5. **Moderate neutrophilia & monocytosis** which reflects chronicity, ex: TRP chronic peritonitis

NB : Findings of hypochloremia, hypokalemia, and alkalosis are also findings in :

- **Distal stenosis**
- **Abomasal displacement**
- **Abomasal impaction**

Vagal indigestion, Houfler’s syndrome, chronic indigestion

Diagnosis

Therapeutic diagnosis

- Most common way
- Depends on **history of no response to treatment & medications** , same complain :no defecation , low milk yield , no rumination , no feed intake, mild distension, bloating
- Same as signs of abomasal displacement, impaction

Tentative diagnosis

- Depends on symptoms , shape of rumen , inspection , palpation , rectal palpation , auscultation

Ultrasonographic

- Detects motility of reticulum
- **Normal motility** : excludes TRP
- In cases of TRP adhesion and impairment in motility.
- detects Hypermotility form

Radiography

- Detects any abscesses , foreign body

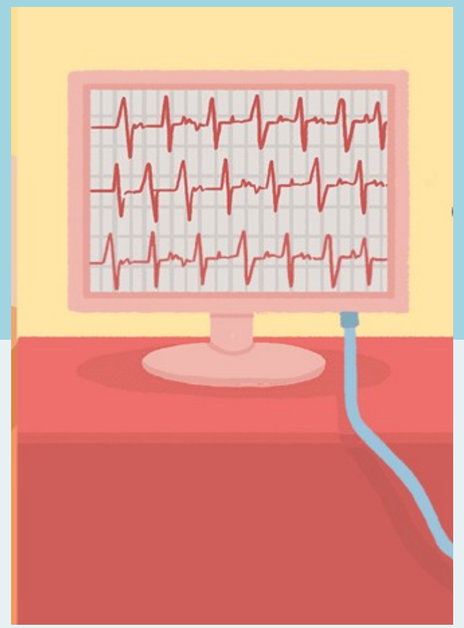
Rumen cl concentration

- Increase chloride usually associated with **abomasal posterior stenosis**

Exploratory laparotomy and & rumenotomy

- Incision to detect distensions and explore reticulomasal opening to see its sensations, contractions

Diagnosis

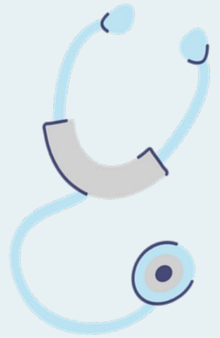


Bradycardia in hypermotility

To detect whether reason of bradycardia is due to vagal nerve affection or no? **atropine test :**
inject atropine slowly (which is Ach antagonist)

⇩

inc heart rate (by >16% than its normal rate, from 50 to 57 bpm) this is
response to S/C of 40 mg atropine sulfate , (0.06 mg/kg)
Or I/V 0.02 mg/kg
This response indicates that vagal nerve is affected & atropine stopped its abnormal tonicity
Sometimes this diagnosis is inaccurate because it depends on degree of tonicity of vagal nerve



Differential diagnosis of chronic indigestion

1. Ileus (paralysis) of the duodenum or jejunum

Detect by ultrasonography that shows motility of intestine

2. Frothy ruminal bloat,

Responds to treatment while vagal indigestion shows no response

3. Abomasal displacement esp. RDA

By clinical signs

4. Abomasal impaction

by clinical signs , **impaction** maybe a sign of TRP ; vagus indigestion that caused **abomasal impaction .**

- **abomasal ulcer** (ulcers cause functional stenosis).

Vagal indigestion, Houfler’s syndrome, chronic indigestion

Conservative treatment

- **vitamin B complex**
- **Ca gluconate** to Inc. tone of muscles and nerves & oral K chloride.
- **ACTH or glucocorticoids** to dec stress and Inc. glucose
- **Fluid** (ringer ONLY (bec lactate will convert to bicarbonate and exacerbates condition), **saline** bec its mild acidic which gives good effect) & **electrolytes** (to treat alkalosis & dehydration) & **mineral oil**
- **Rumen transfaunation**
- Foreign bodies should be removed; abscesses adherent to the reticulum can be drained

Treatment

Usually treatment is hard esp. in posterior stenosis

Some cases

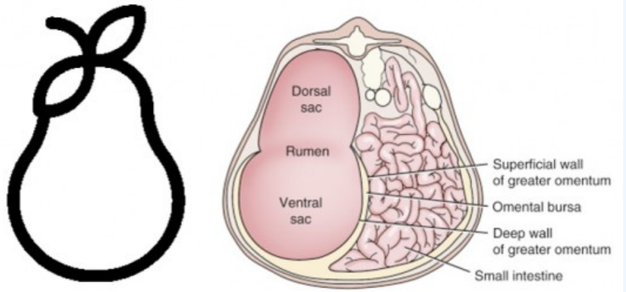
Some cases recovered after birth but may reoccur at next pregnancy
Bec gravid uterus press on ruminal content which inc intensity of adhesions if theyre present

If **no** improvement

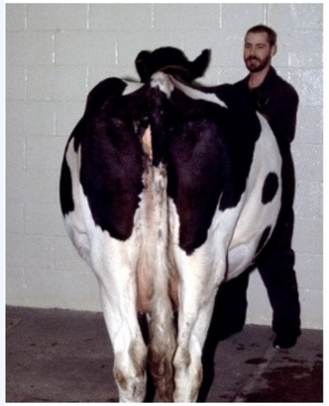
If no response for > 2 wks

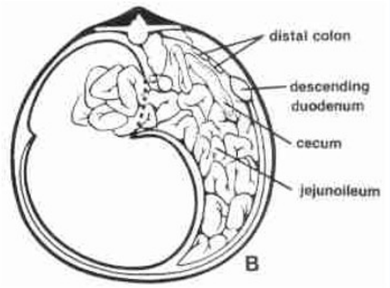
↓

Slaughtering .

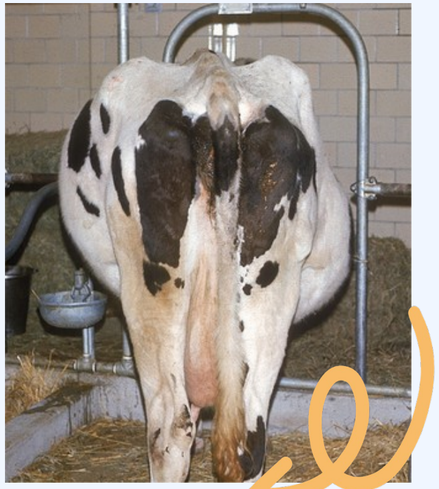


Farm Animal Surgery

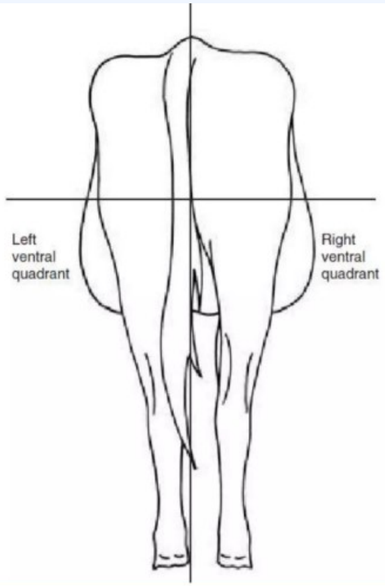




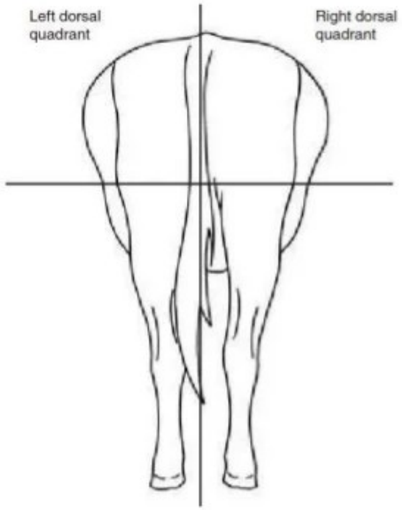
Farm Animal Surgery



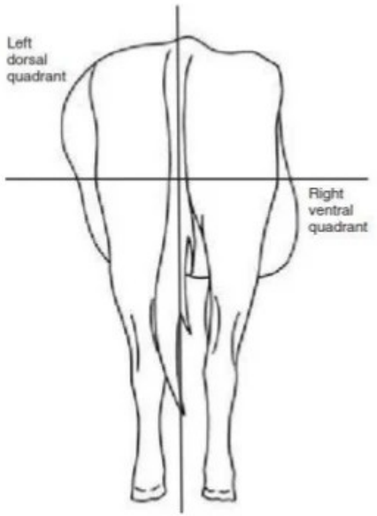
Classic papple shaped abdominal profile of vagal indigestion, with an apple on the left and a pear on the right



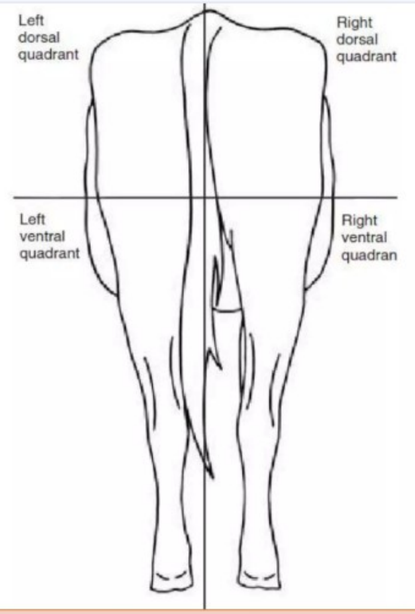
Ascites causing gross distension of the right and left ventral quadrants of the abdomen Posterior view.



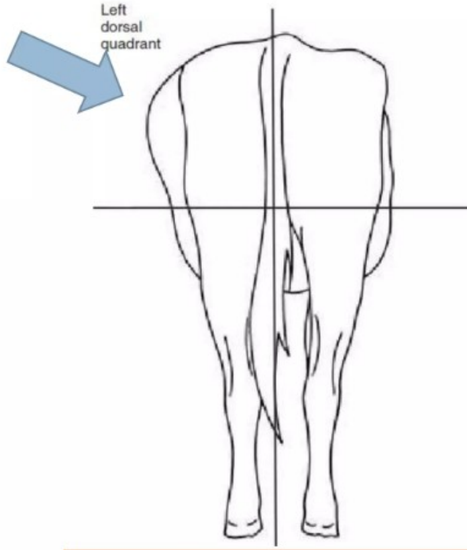
Pneumoperitoneum causing distention of the left and right dorsal quadrants of the abdomen. Posterior view



Vagal indigestion causing distention of the left dorsal and right ventral quadrants of the abdomen. Posterior view.



Normal silhouette of the lateral contours of the abdomen (Posterior view)



Ruminal bloat causing distention of left dorsal quadrant of abdomen(Posterior view)

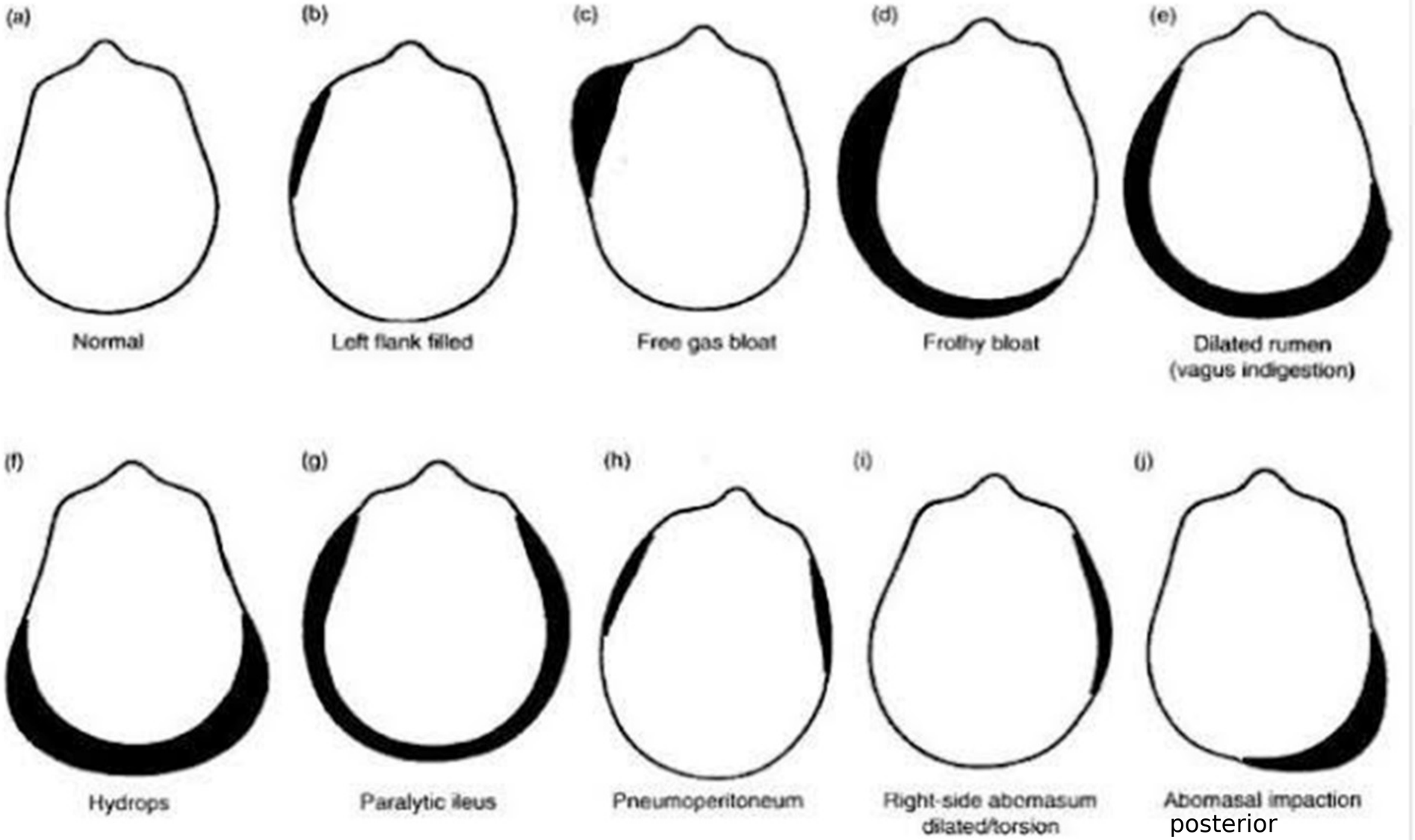
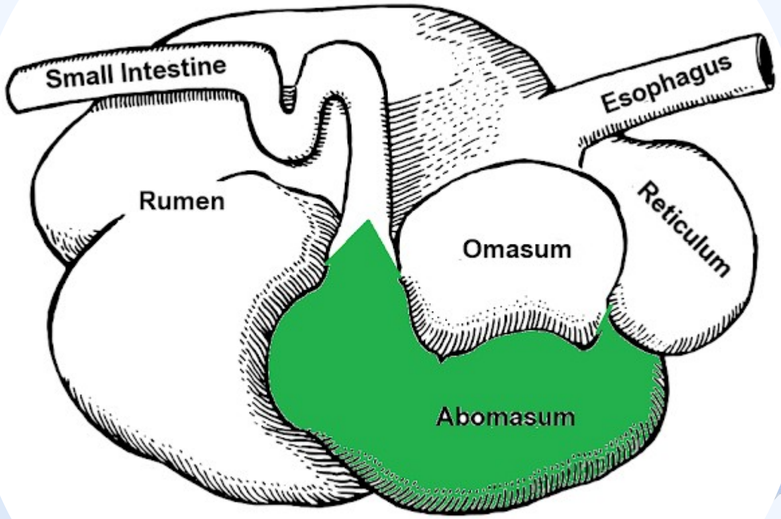


Fig. 6.1 Silhouettes of the contour of the abdomen of cattle, viewed from the rear, with different diseases of the abdominal viscera. (After Stober M, Dirksen G. Bovine Pract 1977; 12:35–38.)

Abomasal diseases

- Abomasal ulcer
- Abomasal impaction
- Abomasal volvulus or torsion from uncorrected RDA
- Right displaced abomasum
- Left displaced abomasum



- These diseases are associated with **management**, environmental, genetic factors.

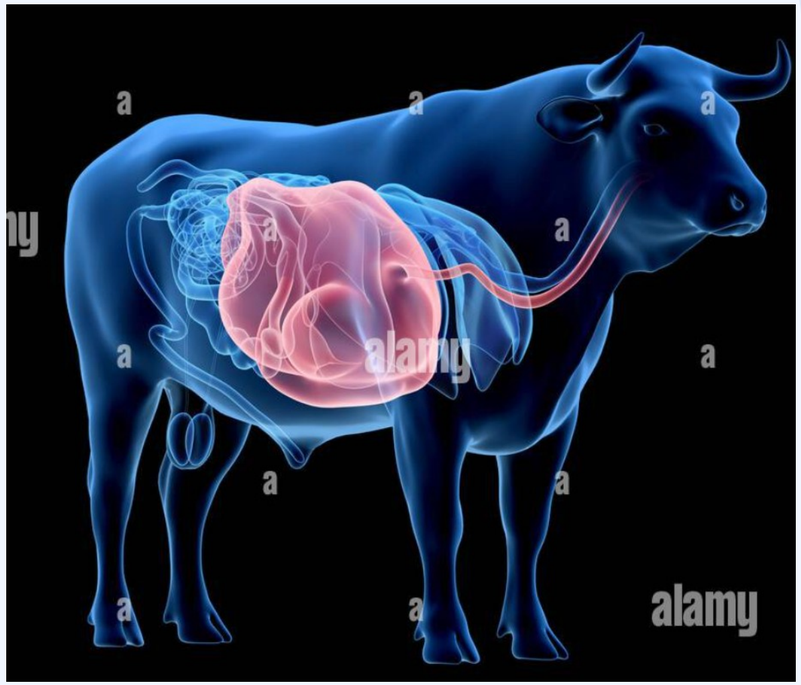
(SARA , frothy feedlot tympani can also result from managemental factors as discussed before)

Dairy cattle are being selected for high milk production & fed **large quantities of grain** with **limited exercise** is the precursor of abomasal hypomotility, impaction & displacements

Abomasal ulcers

Definition

Local or diffuse interruption in continuity of abomasal mucosa, submucosa due to any inflammation



CCC by

- Abdominal pain, anorexia and depression
- Abnormal GIT motility (Diarrhea or constipation)
- Abomasal hemorrhage (melena) characteristic to ulcer, but does not necessarily appear with all types of ulcer, only hemorrhagic type dark tarry , black feces

Due to mixture of HCL + blood acid hematin

- **Peritonitis** if occur rupture of abomasum

Abomasal ulcers

classification

o Signs of ulcer depends on site & type of ulcer

o The most common site is **pyloric** region due to continuous opening and closure of sphincter so food is liable to be trapped

Non perforating ulcer (doesn't break continuity of wall)

- With erosion (Type I ulcer),

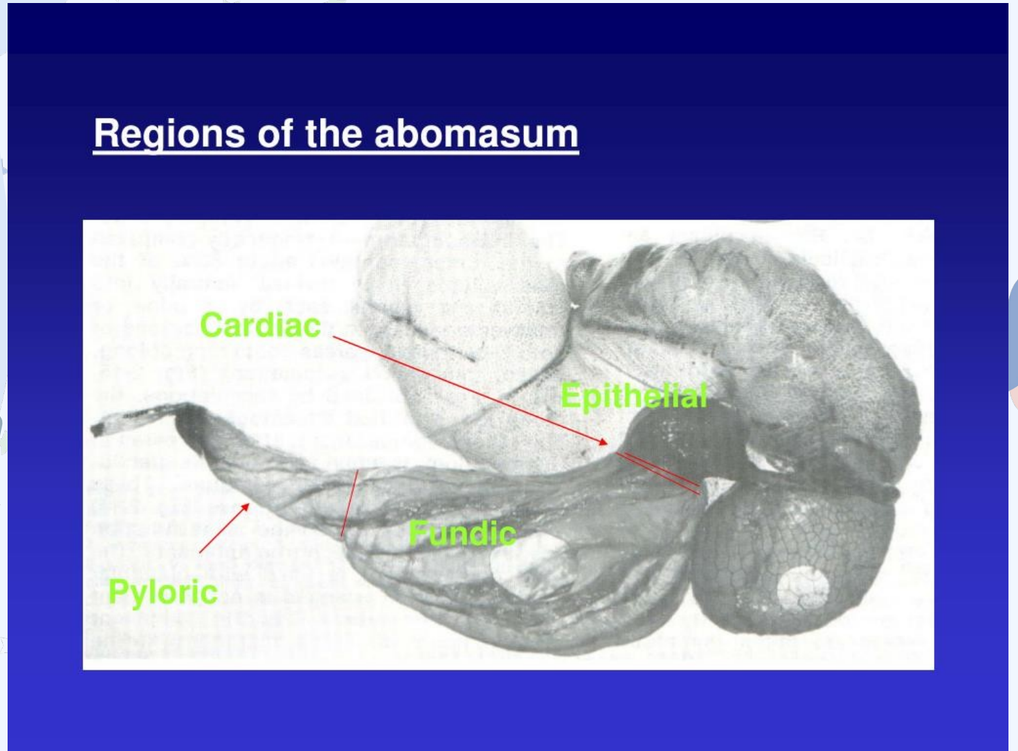
(No signs & detected only at P.M.)

- With **bleeding** in internal mucosa (type II ulcer)

Perforating ulcer

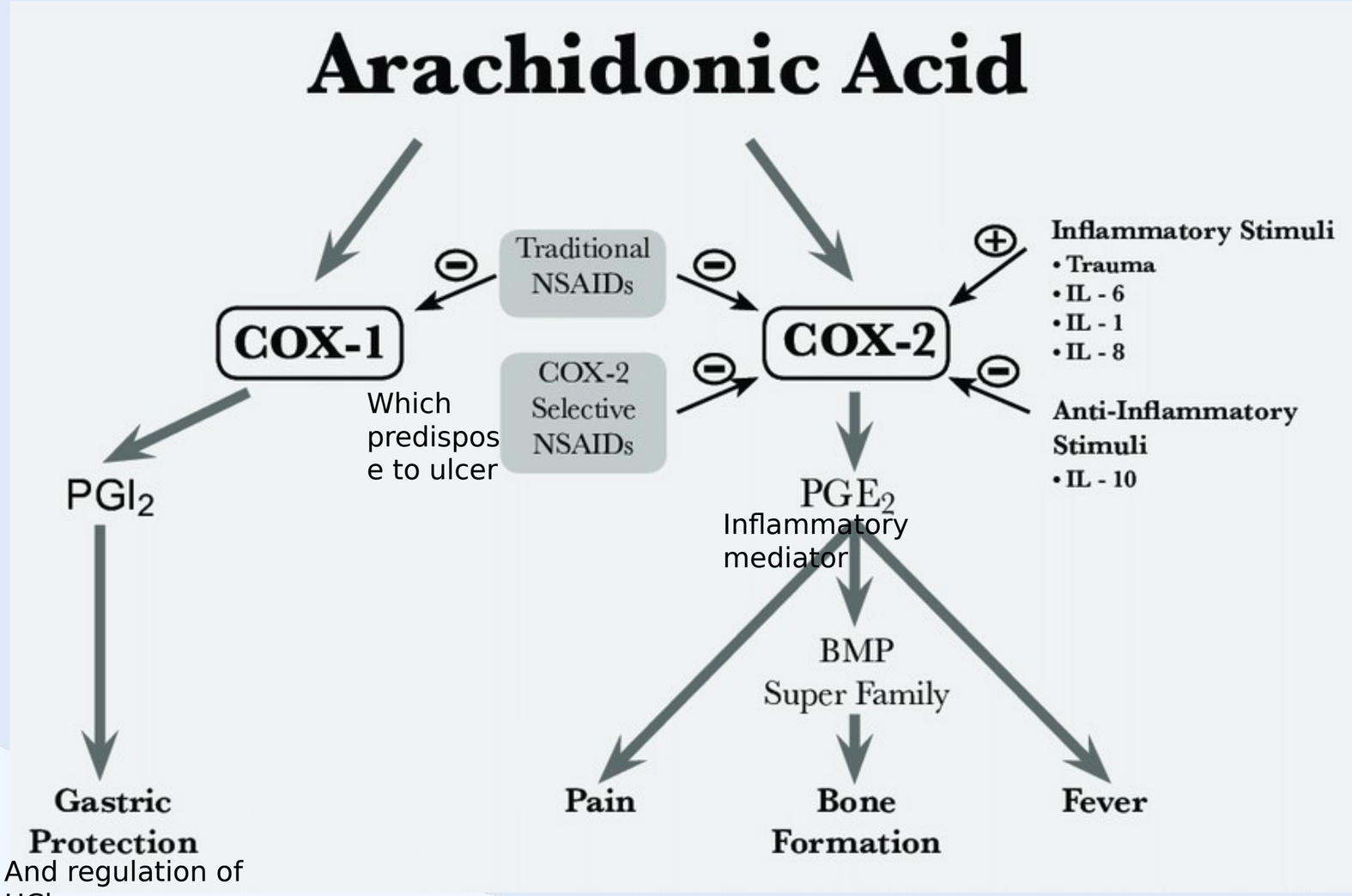
With **local** peritonitis (type III)

With **diffuse** peritonitis (type IV)



NB, for illustration only

COX-1 maintains the normal lining of the stomach and intestines, protects the stomach from digestive juices



Any stress may interrupt this pathway and , ↑ cortisol and so, interrupts PG pathway

Abomasal ulcers ?

Etiology and pathophysiology

1. Nutritional stress

- High concentrates doses for long period VFA , LA which increases acidity affects abomasal wall
- Feeding on coarse low quality roughage for long periods

2. Physical stress

During parturition

Transportation for long periods

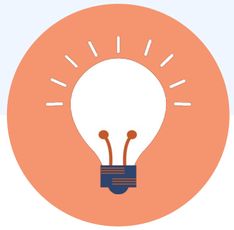
3. Toxicities as nitrates, arsenic

4. Chemical stress

- NSAIDs
- Corrosive substance
- Any chemicals ingestion
- Bile salts in intestinal reflux which is very corrosive

5. Secondary (reticular & abomasal diseases ex TRP , impaction , torsion,

- **Infectious :**
- Bacterial : clostridium perfirnges. Strept
- Viral : bvd , rinderpest, lumpy skin disease in late stages
- Parasitic : mycotic infections after chronic acidosis
- Miscellaneous lover , kidney affections , inc in toxins



PGE2 responsible for :

- preservation of mucosal integrity,
- mucous secretion which protects it from HCL, pepsin
- microcirculation & decrease HCL secretion.

In addition to **gastrin** hormone which is responsible for

- renew , integrity ,
- regulation of HCL secretion Gastrin secretes hcl in presence of food and vagus tone & so , any ↓ in HCL levels initiates high sec of gastrin hormone .

o Any stress factors inc.

Abomasal ulcers

Clinical signs

Non-perforating ulcer

(type I) (with erosion)

Anorexia & signs of abdominal pain as kicking of abdomen.

(type II) (with bleeding)

1. **Anorexia** & signs of abdominal pain as kicking of abdomen in addition to :
2. **Melena**(black feces) (intermittent acc to amount of bleeding) & positive fecal occult blood test
3. **Anemia** with pale eyelids, with history of milk production & depression & appetite
4. **Inc** in gastrin level if measured indicates bleeding ulcer bec bleeding dilutes acidity of abomasum initiates gastrin secretion

Perforating ulcer

Type III (Localized peritonitis)

Signs as in TRP, It may be followed by invasion of m.o

- Anorexia, drop in milk yield
- Abdominal pain esp.at palpation of right ventral abd. Wall
- Dec ruminal motility or absent with mild bloat
- Fluctuating fever with inc pulse &inc respiratory rate.

TYPE IV (Diffuse peritonitis)

- More toxic as it causes toxemia , more severe than localized
- Abruptly dec milk production with ruminal stasis
- Abdominal pain & reluctant to move with audible grunt or groan with each breath
- **Temperature**: Rapid inc followed by sudden drop of temperature
- **Respiration:** shallow respiration,
- **Heart rate:** Prominent tachycardia
- Severe dehydration & weakness
- Recumbency, cold extremities, shock & coma → death within 4-48 hrs

Abomasal ulcers
Treatment

If a oral medication has to be provided & must be guided to abomasum and not rumen , closure of esophageal groove is necessary by :
vasopressin hormone or 60 ml cuso4 5% or Sodium bicarb 10%
In sec closure of groove for only 1 minute ay which medication is provided immediately

1. Fluid & electrolyte therapy

To treat anorexia, bleeding, anemia

2. Blood transfusions in hemorrhaging

To treat ulcers and hemorrhage

3. Broad spectrum antibiotics

can help when an animal has signs of Peritonitis

4. **Oral** administration of antacid agents

Mg hydroxide, Aluminum hydroxide (Maalox), (Gaviscon).
Binds with bile salts and neutralizes Hcl but its only problem short acting

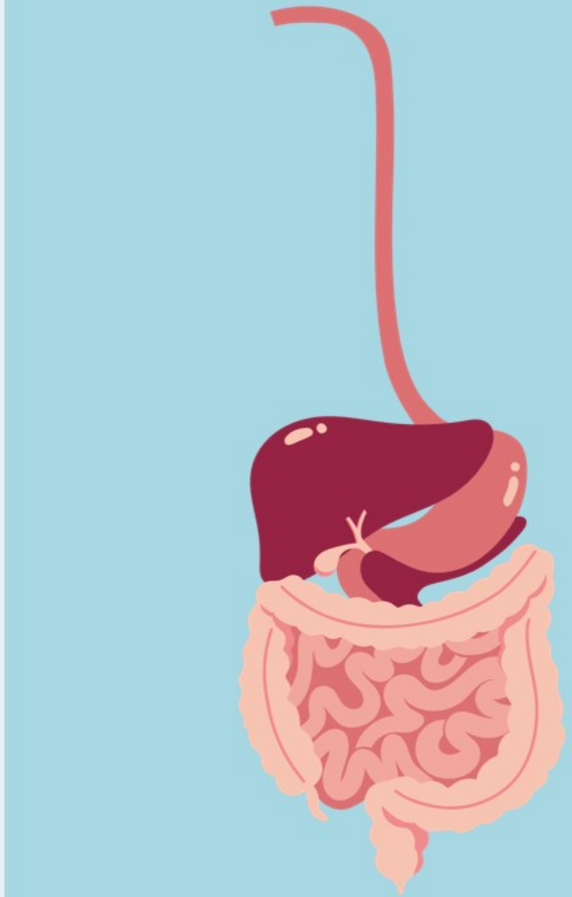
5. H2 blockers

Ranitidine ,famotidine, Zantac, Cemitidine
They work by blocking histamine at the H2 receptors of the parietal cells, which decreases acid production.
Oral and injection
These medications are useful for calves but not for **large ruminants** as its very hard for medications to reach abomasum , it's directed to rumen so any medication is not given to ruminants orally
and if provided by **injection** of h2 blocker will provide a short action for 2,3 hrs only in large animals, must be repeated for 5,6 times and its expensive so it's replaced by : Omeprazole

6. Omeprazole

Proton pump inhibitors.
medications that cause a profound and prolonged reduction of stomach acid production.
They do so by irreversibly inhibiting the stomach's H /K ATPase proton pump, can be used for calves (pre ruminant)
In large animals injection only They provide good relief in first day then its effect decreases in second, third day so administrated only for 1 day and not

Dietary abomasal impaction in cattle



Other face of posterior stenosis after vagal indigestion, it’s a sequalae to TRP

Etiology

1

- Excessive consumption of poor-quality indigestible roughages
- Large quantities of chopped roughages or short roughages → doesn't stay enough in rumen and goes to abomasum

2

- Sand which is precipitated in rumen ,abomasum →affects motility
- foreign bodies

3

- Inadequate mineral supplementation leads to pica
- Restriction of water potentiate the previous causes

4

Abomasal hypomotility after parturition

5

vagal indigestion esp. ventral branch (posterior stenosis)(achalasia in pyloric opening)

6

Abomasal ulcers which affects motility

NB : history of :

Transportation for long time with signs of kicking , discomfort , excessive movement of animal providing that animal didn't suffer from anything with owner , by reaching your farm → inappetence.

This indicates a case of :

TRP non penetrating then became penetrating.

The case was treated , then complication of chronic peritonitis occurred with signs of chronic indigestion and toxemia , the case was treated , then after long time , →signs of abomasum discomfort and bulging at lower left flank

Pathogenesis

- **Continuous secretion of HCL**
- **Continuous accumulation of fluids & ingesta within abomasum Due to relaxation of abomasal muscles along with distension**
- **Abomasal reflux → to rumen with ↑ cl conc.**
- **Sub-acute obstruction of upper GIT with feces**
- **Dehydration develops with electrolyte imbalance**
  (hypochloremic,hypokalemic metabolic alkalosis)

impaction of abomasum may be severe enough to cause irreversible abomasal atony (hopeless case with unresponsive treatment

History of animal inappetence with mild frothy tympany and soft scanty defecation, or hard pelleted feces or dark feces → due to bleeding
Foul odor . History of vomiting

Anterior and posterior stenosis could be differentiated by cl ions conc in rumen

Anterior	posterior
Normal cl	Inc cl due to reflux of content .

Clinical findings

As Posterior stenosis

- ↑ heart rate >120 bpm→ INDICATES HOPELESS CASE
- ↑ Respiratory rate & expiratory grunt due to abdominal distension

Marked dehydration due to alkalosis

Acute diffuse peritonitis, shock & death are observed when abom. rupture occur in severe cases

Differentiation of RDA from abomasal impaction:
Abomasal impaction : R lower area
RDA : upper area

Complete anorexia, dec In MY , recurrent mild frothy tympani, scanty feces (black, pale, hard pellets, soft , mucus)

Auscultation : rumen stasis (no motility heard)

Inspection : at lower R flank region : late stage → filled with fluid , upon direct palpation abomasum is felt which is normally not felt upon rectal palpation

Deep palpation & strong percussion in area of distension : animal expresses grunting due to stretching of abomasal wall . In cases of posterior stenosis due to TRP → pain upon palpation of sternum

In sand impaction →weight loss with chronic diarrhea with sand in feces -weakness, recumbency & death within few weeks.

Agitation, ballottement with auscultation at R lower flank : fluid splashing sound (due to accumulation of feed with gases)

Treatment

1. Fluid therapy & electrolytes (saline & ringer) to treat **alkalosis**

2. isotonic acidifiers orally(aluminum chloride, potassium chloride 120 gm to ↓ alkalinity of abomasum &to regulate electrolytes and acid base imbalance (source of K & cl)

3. Laxatives & lubricants as DSS (dioctyl sodium sulphosuccinate , Paraffin ,linseed oil)

4. Ca preparation to improve tonicity of GIT

5. Prokinetic drugs (parasympathomimetic) : drugs used to enhance GIT & abomasal emptying rate or (Macrolides) → erythromycin 8-10 mg/kg i/v but unavailable injection or (ivermectin : 200 microgm / kg i/v but i/v is of high risk)

6. H2 blockers if melena present to↓ hcl

7. Surgical interference.

Diagnosis

Confirm diagnosis by :

1. PH of blood : acid base imbalance) → hypokalemic, Hypochloremic metabolic alkalosis
2. ↑ Cl conc in ruminal fluid > 20 mmol/l → 40 mmol/l
3. Auscultation with agitation at lower R flank → splashing sound

Abomasal reflux

1

Definition

Reflux of abomasal fluids into the omasum, reticulum & rumen, occur when the abomasal fluid fail to move normally through the pylorus into small intestine

2

Etiology

- LDA or RDA
- Abomasal impaction
- Vagus indigestion(posterior stenosis)
- Peritonitis, Toxemia
- Compression of abdomen as in advanced preg. & Intussusception

3

Characterized by

- Hypochloremic, hypokalemic metabolic alkalosis
- Inc ruminal chloride , ↓ in cl blood conc
- ↓ ruminal buffering capacity

4

Treatment

Remove the excess fluid by stomach tube
Parenteral electrolyte